Alcohol and sleep apnea

Victoria S. Rains MD LTC MC USAR*
Thomas F. Ditzler PhD**
Richard D. Newsome PhD Maj BSC USAF**
Sonia Lee-Gushi RPSGT CRTT†
Edward J. Morgan MD PhD††

Acute ingestion of ethanol induces vasodilation and swelling of respiratory mucosa; it depresses respiratory centers resulting in hypotonia of oropharyngeal dilator muscles and inducing or aggravating sleep apnea. However, no association between the sleep apnea syndrome (SAS) and Alcohol Use Disorders (AUD) has been demonstrated.

Introduction

Sixty eight patients diagnosed as having SAS by polysomnography over a 6-month period were evaluated for the presence of AUD using the Michigan Alcoholism Screening Test (MAST) and an alcohol consumption history questionnaire. The prevalence of AUD was obtained by including both the positive MASTs and/or with admitted hazardous consumption known to correlate with significant physiologic risk (5 or more standard drinks per day on a regular basis). The correlation between moderate and severe SAS known to be associated with increased risk for mortality (Apnea Index > 20), was then evaluated with respect to presence or absence of AUD.

The prevalence of AUD in the SAS patients as defined by MAST alone was 22% (15/68), a figure which correlates with the prevalence found in other studies of in- and outpatient cohorts. Twelve sleep apnea patients (18%) admitted that they consumed hazardous amounts of alcohol. The prevalence of AUD, using a positive MAST and/or admitted hazardous consumption, was 31% (21/68). Eighty six percent (18/21) of the patients with AUD were found to have an apnea index of 20 or greater, indicating a high mortality risk. AUD and high mortality risk sleep apnea were in significant correlation ($N = 68, r = 0.24, p < 0.05$).

Our study demonstrates a significant correlation between AUD and SAS, even in the absence of acute alcohol ingestion immediately prior to the polysomnographic test. In addition, the correlation between AUD and SAS severity suggests that the morbidity seen in both AUD and SAS may be related to the same pathophysiologic mechanism.

Background

The effect of alcohol on sleep is well known. Acute alcohol ingestion disrupts the normal sleep pattern by reducing sleep latency and increasing wakefulness in the last half of the night[1]. Abnormal sleep patterns occur in alcoholics, even after prolonged sobriety, because of the effects of chronic alcohol toxicity on sleep centers[2,3].

The relationship between acute alcohol ingestion and sleep apnea has also been investigated. Taasan et al demonstrated that 2 ml/kg of alcohol consumed 1 hour prior to bedtime induced apneic episodes (cessation of breathing at the nose and mouth for 10 seconds or more[4]) in normal subjects[5]. Scrima and colleagues found similar results in both normal subjects, as well as in individuals diagnosed with the SAS after 3 oz of 80-proof alcohol[6]. Alcohol induces apnea through both central and peripheral mechanisms. Acute ingestion of ethanol induces vasodilation and swelling of respiratory mucosa and depresses respiratory centers, resulting in hypotonia of oropharyngeal dilator muscles and inducing or aggravating sleep apnea[7,8]. Peripherally, alcohol causes vasodilation resulting in pharyngeal edema[7].

There is also a possible link between chronic heavy alcohol ingestion and sleep apnea. Vitiello and colleagues demonstrated a high prevalence of nocturnal hypoxic episodes in a population of recovering alcoholics[8]. In addition, the severity of the alcohol abuse was found to correlate with the level of nocturnal hypoxia[9,10]. It was suggested that the mechanism was sleep

* University of Hawaii, John A. Burns School of Medicine
Division of Geriatric Medicine and
Tripler Army Medical Center Augmentation Unit, US Army Reserve
** Tri-Services Alcohol Recovery Facility
† Sleep Disorders Center, Kuakini Medical Center
†† University of Hawaii, John A. Burns School of Medicine
Department of Medicine and
Kuakini Medical Center, Pulmonary Services

Please address correspondence and request for reprints to:
Victoria S. Rains MD
Division of Geriatric Medicine
347 N Kuakini St, 9th Floor HPM
Honolulu, Hawaii 96817

Article received December 19, 1991.

apnea, however these patients were not tested by polysomnography.

The relationship between chronic alcohol use and the SAS (5 or more apneic episodes per hour of sleep[4]) is important because both diseases result in significant morbidity. In fact, the sequelae of both diseases include hypertension, arrhythmias, depressive symptoms, cardiac disease, cerebrovascular accidents and, most importantly, death. He et al demonstrated that sleep apnea patients with an apnea index (AI) > 20 had significantly greater mortality than those with an AI < 20[11]. The correlation between SAS severity and the mortality in alcoholic SAS patients has not been studied.

Our study investigates 2 aspects: (1) What is the prevalence of AUD in patients diagnosed with SAS by polysomnography? and (2) is AUD related to the severity of SAS? The term "Alcohol Use Disorder" (AUD) was chosen to conform with the criteria for alcohol abuse and/or alcohol dependence found in the third edition of the Diagnostic and Statistical Manual of the American Psychiatric Association (DSM III-R)[12].

Methods

Sixty eight patients presenting to the Kuakini Medical Center Sleep Laboratory for polysomnography over a 6-month period were diagnosed as having SAS. These patients were evaluated for a possible associated AUD, using the Michigan Alcoholism Screening Test (MAST). MAST is a weighted, 25-item questionnaire, which screens for the presence of alcoholism when 5 or more points are calculated on the basis of positive responses[13].

The results of MAST were then correlated with the alcohol consumption history obtained from the sleep laboratory questionnaire developed by Laughton Miles[14]. The alcohol consumption questions solicit information concerning the type of alcoholic beverage consumed and the pattern of use as measured by both quantity and time frame. Individuals with admitted consumption of 5 or more standard drinks on a regular basis (daily or on weekends) were considered to have a possible AUD because Sanchez-Craig and colleagues demonstrated that this pattern of "hazardous consumption" results in a significant morbid risk to individuals[15].

Finally, the relationship between the severity of AUD and of SAS was examined by comparing MAST results and the presence or absence of hazardous consumption of alcohol. The severity of SAS was defined by an apnea index of < 20 versus ≥ 20. An apnea index of 20 is thought to require treatment because of the increased risk of death as a consequence. Such patients are generally considered to be candidates for treatment with continuous positive airway pressure because it has been demonstrated that the incidence of mortality is reduced[11].

Results

The average age of patients referred to the sleep laboratory was 48 years, with a range of 20 to 73 years. The prevalence of MAST positive sleep apnea patients was 22% (15/68). None of the 12 female patients were screened positive on MAST, whereas 15 of 56 males were MAST positive for a prevalence of 27%. Fourteen individuals evaluated by the sleep laboratory did not complete MAST; the participation rate was 85%, therefore.

TABLE 1

Relationship between admitted hazardous consumption and MAST scores

		POS. MAST	NEG. MAST	TOTAL
HAZARDOUS CONSUMPTION* (Number of Patients)	Present	6	6	12
	Absent	9	47	56

* Hazardous consumption is the admitted consumption of 5 or more standard drinks on a regular (daily or on weekends) basis.

TABLE 2

Relationship between alcohol use disorders and sleep apnea syndrome

	SEVERITY OF SAS			AGE
	Apnea Index <20 (No. of Pts.)	Apnea Index >20 (No. of Pts.)	Total No.	Mean (SD**)
AUD* Present	3	18	21	46.8 (7.7)
AUD* Absent	18	29	47	48.5 (12.8)

* Patients with Alcohol Use Disorders (AUD) are MAST positive and/or admit to Hazardous Consumption.
**Standard Deviation

Twelve sleep apnea patients (18%) admitted to regular hazardous consumption of alcohol. Six of the 15 MAST-positive patients did not complete the alcohol consumption questionnaire. The correlation between MAST scores and reports of hazardous consumption was significant ($N = 68$, $r = .27$, $p < 0.05$), as shown in Table 1.

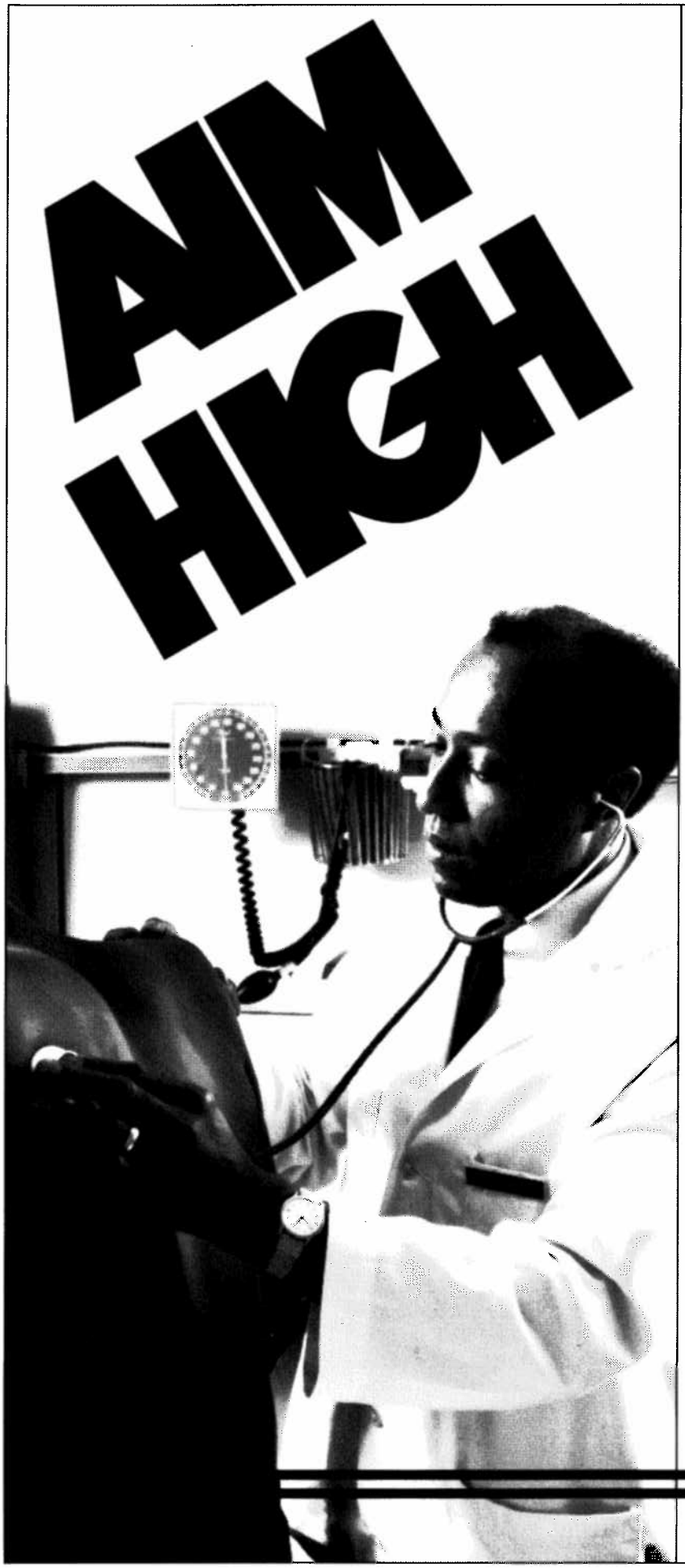
Since our study addressed the impact of both chronic alcohol abuse and dependence on sleep apnea, the number of SAS patients with positive MASTs and/or hazardous consumption were combined. This produced a combined prevalence of 31% (21/68). The combined prevalence in males was even higher 38% (21/56); none of the females screened positive on MAST or indicated hazardous consumption.

Using an Apnea Index (AI) of 20 as a cut-off score to evaluate risk for mortality, 86% (18/21) of the patients with AUD were found to have a high mortality risk related to their sleep apnea (Table 2). AUD and high mortality risk sleep apnea were significantly correlated ($N = 68$, $r = 0.24$, $p < 0.05$).

Discussion

The prevalence of AUD in the SAS population as determined by MAST was 22%, a figure which compares with the

(Continued on page 285) ➤



SPECIALIZE IN AIR FORCE MEDICINE.

ER Physicians. Radiologists. OB/GYNs and other specialists! Today's Air Force gives you the freedom to specialize without the financial overhead of running a private practice. Talk to an Air Force medical program manager about the tremendous benefits of becoming an Air Force medical officer:

- No office overhead
- Dedicated, professional staff
- Quality lifestyle and benefits
- 30 days vacation with pay each year

Examine your future in the Air Force. Learn if you qualify. Call

USAF HEALTH PROFESSIONS
COLLECT
(808) 538-0805

AIR FORCE

prevalence found in other studies of in- and outpatient medical populations[16,17,18]. However, MAST did not identify 9% (6/68) of patients with admitted hazardous consumption. The definition of AUD in the DSM III-R includes individuals who knowingly consume alcohol in the face of medical problems exacerbated by the consumption[12]. In the sleep laboratory, SAS patients with hazardous consumption would fit this definition. Although there was a significant correlation between MAST and the presence or absence of hazardous consumption, the 9% false negative rate was greater than in a study by Cyr and Wartman, who found that in an ambulatory practice population only 7 of 242 individuals were heavy drinkers who did not screen positive on MAST[19].

MAST might not identify certain hazardous drinkers because the weighted items on MAST generally screen for social consequences of alcoholism, which not all alcoholics develop. The medical consequences that are picked up by MAST include only cirrhosis or an identification by physicians that alcohol is the etiology of medical complaints. However, previous studies have demonstrated low physician recognition of alcoholism[17,18,20]. Since the medical consequences of alcoholism and sleep apnea (sleep disturbance, depressive symptoms, hypertension, arrhythmias, etc) are almost identical, symptoms may not have been attributed to the presence of AUD.

Our study also found that 40% of MAST positive patients did not report hazardous consumption. This discrepancy is most likely related to the sleep laboratory policy of advising SAS patients to discontinue drinking. Patients referred to the sleep laboratory are generally quite symptomatic with their sleep disturbance and are routinely informed that alcohol can worsen their symptoms. In addition, physicians who refer patients for sleep studies commonly give the patient the same caveat because of the association between alcohol ingestion and apneic events. Thus, at the time the alcohol consumption portion of the sleep questionnaire was completed, it was unlikely that patients would report hazardous consumption. Lee and DeFrank point out that MAST may not be an accurate reflection of current alcohol consumption and may misidentify a person who no longer consumes alcohol[21]. However, since our study evaluates the possible toxic effect of chronic alcohol consumption, MAST is a useful tool to identify subjects who may have had a past history of hazardous drinking.

To get a more accurate indication of the prevalence of AUD in SAS patients, it is necessary to add the number of MAST positive patients to the number of MAST negative patients who indicated hazardous consumption. This yields a combined prevalence of 31%, 38% in males alone. The high prevalence in males is important because both AUD and sleep apnea are predominantly male diseases.

The 31% combined prevalence of AUD in SAS patients found in this study suggests an association between AUD and sleep apnea. This association cannot be explained on the basis of acute ingestion of alcohol because patients at the sleep laboratory who ingested alcohol prior to the visit were generally not evaluated by polysomnography. In addition, sleep apnea patients with AUD were more likely to have an AI of > 20, indicating there may be an association between AUD and the severity of SAS.

In our study, a sub-sample of 17 of the 68 SAS identified 8 with AUD. None of the 8 had been referred by their physicians for alcohol counseling, although 4 had discontinued or cut down their drinking. As in other studies demonstrating low rates of recognition by physicians and referral for alcoholism treatment[17,18,20], the concern is that complete evaluation of SAS patients should include screening and referral for AUD. This is because acute alcohol ingestion aggravates SAS and patients with AUD are unlikely to quit drinking for prolonged periods without the help of a recovery program. The results of our study suggest that patients with AUD are more likely to present with an AI of 20 or more; He et al demonstrated this degree of severity carries a significant risk for mortality[11]. Therefore, intervention in AUD is as important as therapy in sleep apnea.

The 31% combined prevalence of AUD in SAS patients and the likelihood of having an AI of 20 or more, suggests that the toxicity of chronic alcohol ingestion plays a role in the development of morbidity and mortality in sleep apnea. As a corollary, the morbidity associated with alcoholism may be attributed in part to the medical sequelae of sleep apnea (arrhythmias, hypertension, etc), especially since these are frequently found in alcoholic patients. This raises the question whether sleep apnea may be a common but undiagnosed problem in chronic alcoholics. The finding by Vitiello and colleagues of nocturnal hypoxia in abstaining alcoholics suggests this to be the case[9,10]. Further studies are needed in order to evaluate the association between alcoholism and sleep apnea.

REFERENCES

1. Pokorny AD: Sleep Disturbances, Alcohol, and Alcoholism: A Review. In: Williams RL and Karacan I. eds. Sleep Disorders Diagnosis and Treatment. New York: John Wiley and Sons, 1978: 233-259.
2. Adamson J, Burdick JA: Sleep of Dry Alcoholics. *Arch Gen Psychiatry* 1973;28:146-149.
3. Snyder S, Karacan I: Sleep Patterns of Chronic Sober Alcoholics. *Neuropsychobiology* 1985;13:97-100.
4. Taasan VC, Block AJ, Boysen PG, et al: Alcohol Increases Sleep Apnea and Oxygen Desaturation in Asymptomatic Men. *Am J Med* 1981;71:240-245.
5. Scrima L, Broudy M, Nay KN, et al: Increased Severity of Obstructive Sleep Apnea After Bedtime Alcohol Ingestion: Diagnostic Potential and Proposed Mechanism of Action. *Sleep* 1982;4:318-328.
6. Robinson RW, White DP, Zwillich CW: Moderate Alcohol Ingestion Increases Upper Airway Resistance in Normal Subjects. *Am Rev Respir Dis* 1985;132:1238-1241.
7. Remmers JE: Obstructive Sleep Apnea — A Common Disorder Exacerbated by Alcohol. *Am Rev Respir Dis* 1984;130:153-155.
8. Vitiello MV, Prinz PN, Personius JP, et al: Nighttime Hypoxia is Increased in Abstaining Chronic Alcoholic Men. *Alc Clin Exp Res* 1990;14:38-41.
9. Vitiello MV, Prinz, Perrsonius JP, et al: Relationship of Alcohol Abuse History to Nighttime Hypoxemia in Abstaining Chronic Alcoholic Men. *J Stud Alcohol* 1990;51:29-33.
10. Association of Professional Sleep Societies. Diagnostic Classification of Sleep and Arousal Disorders. Rochester 1987:65-69.
11. He J, Kryger MH, Sorick FJ, et al: Mortality and Apnea Index in Obstructive Sleep Apnea — Experience in 385 Male Patients. *Chest* 1988;94:9-14.
12. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition-Revised. Washington D.C. 1987.
13. Selzer ML: The Michigan Alcoholism Screening Test: The quest for a new diagnostic instrument. *Am J Psychiatry* 1971;127: 1653-1658.
14. Miles L: Appendix I — Sleep Questionnaire and Assessment of Wakefulness (SQAW). In: Guilleminault C, ed. Sleeping and Waking Disorders:

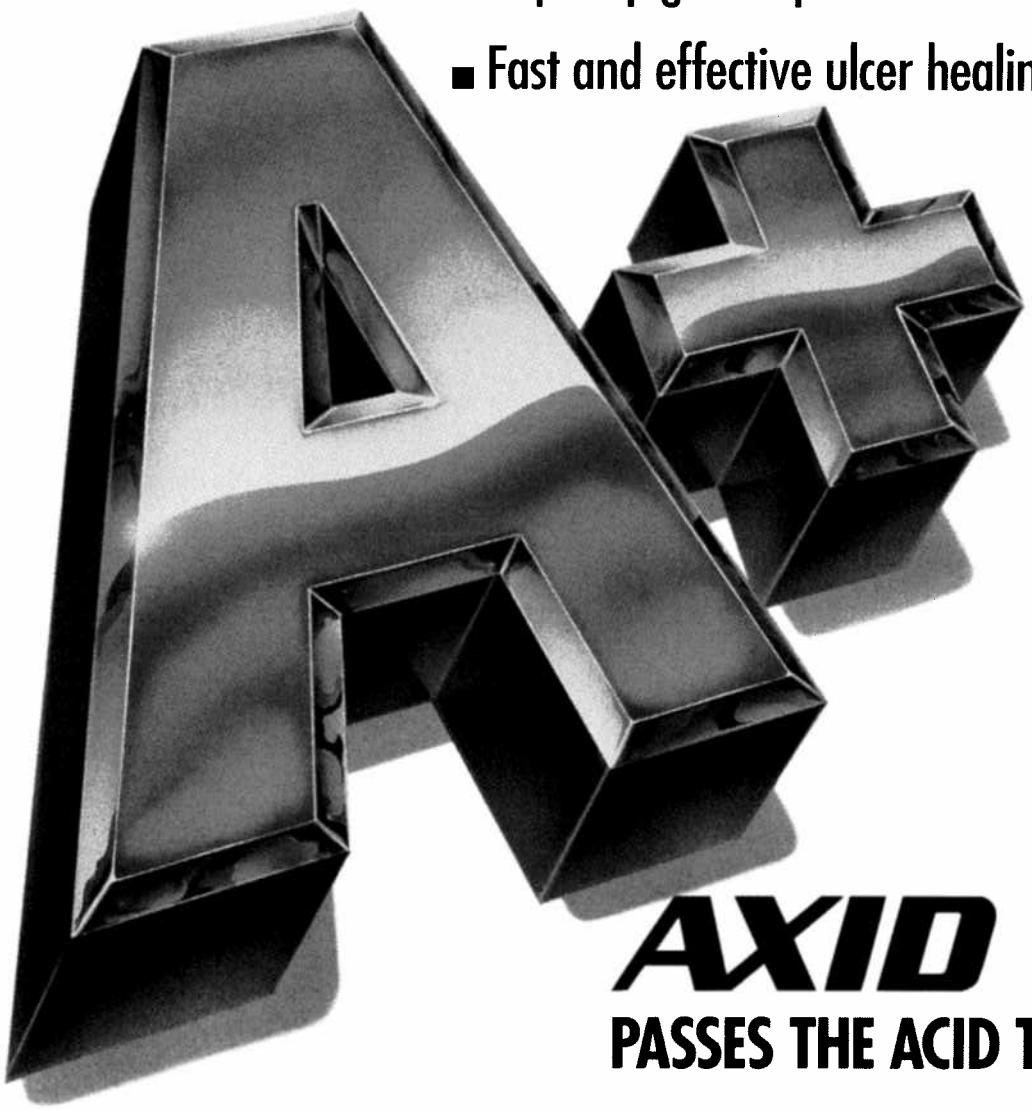
(Continued on page 287) ➤

For excellent response in the treatment of duodenal ulcers...

AXID® nizatidine

has the right answers

- Rapid epigastric pain relief[1,2*]
- Fast and effective ulcer healing[2,3,4]



AXID

PASSES THE ACID TEST

*Most patients experience pain relief with the first dose.

See adjacent page for references and brief summary of prescribing information.

NZ-2943-B-149347

ALCOHOL *(Continued from page 285)*

Indications and Techniques. Menlo Park: Addison-Wesley Publishing, 1982: 383-413.
15. Sanchez-Craig M: Level of Alcohol Use for the Onset of Early-Stage Problem Drinking. In: Chang NC, Chao HM, eds. Early Identification of Alcohol Abuse, Proceeding of a Workship sponsored by the National Institute on Alcohol Abuse and Alcoholism in Cooperation with the World Health Organization and the Pan American Health Organization. 1985:339-347.
16. Yersin B, Trisconi Y, Paccaud F, et al: Accuracy of the Michigan Alcoholism Screening Test for Screening of Alcoholism in Patients of a Medical Department. *Arch Intern Med* 1989;149:2071-2074.
17. Cleary PD, Miller M, Bush BT, et al: Prevalence and Recognition of Alcohol Abuse in a Primary Care Population. *Am J Med* 1988;85:466-471.
18. Moore RD, Bone LR, Geller G, et al: Prevalence, Detection, and Treatment of Alcoholism in Hospitalized Patients. *JAMA* 1989;261:403-407.
19. Cyr MG, Wartman SA: The Effectiveness of Routine Screening Questions in the Detection of Alcoholism. *JAMA* 1988;259: 51-54.
20. Coulehan JL, Zettler-Segal M, et. al.: Recognition of Alcoholism and Substance Abuse in Primary Care Patients. *Arch Intern Med* 1987;147:349-352.
21. Lee DJ, DeFrank RS: Interrelationships Among Self-Reported Alcohol Intake, Physiological Indices and Alcoholism Screening Measures. *J Stud Alcohol* 1988;49:532-537.

■

RBRVS *(Continued from page 279)*

worse off. No one can say that change won't be coming. Those who don't like the RBRVS and the limits on balance billing should consider the alternatives: mandatory assignment, MD-DRGs, and fees set by the government without any professional input. The RBRVS gives the profession a voice, and it enables us to ward off more objectionable measures.

Finally and most importantly, *the RBRVS is good for our patients.* It will increase the emphasis on preventive care and on evaluating and managing their treatment, and decrease the emphasis on costly high-tech services. It also will help improve access to care in underserved rural areas.

So you see, to paraphrase Mark Twain, reports of the death of the RBRVS are greatly exaggerated. But medicine can't rely on trust that everything will turn out okay. We must fight to preserve the promise of physician payment reform.

That means opposing policies that will undermine the RBRVS (such as a behavioral assumption that would lower the fee schedule conversion factor). It means working to change policies — such as the ban on reimbursement for EKG interpretation — that give with one hand and take away with the other. And it means supporting further changes that will make the system even better.

The RBRVS unites physicians under one fair and rational payment system to fight future detrimental budget cuts in Medicare. Lawmakers faced with a divided house of medicine easily can use that division to cut Medicare payments even further. But if they're faced with a profession that's united under the RBRVS, it won't be easy.

Support for the RBRVS has been right — for our profession and for our patients. The RBRVS will protect undervalued evaluation and management services in an era of Medicare budget cutting, increase access and the emphasis on preventive care for patients, and introduce fairness into the Medicare payment system. *But we must fight together — as a profession — to make sure it is implemented in the way Congress intended.*

■